

What is the relationship between dietary potassium intake and blood pressure in adults?

Conclusion

Considerable evidence has demonstrated that a higher intake of potassium is associated with lower blood pressure in adults.

Grade: Strong

Overall strength of the available supporting evidence: Strong; Moderate; Limited; Expert Opinion Only; Grade not assignable For additional information regarding how to interpret grades [click here](#).

Evidence Summary Overview

A total of 10 articles met the inclusion criteria and were reviewed. Of the 10 articles, five were systematic reviews/meta-analyses, four were randomized controlled trials (RCTs) and one was a three period, non-randomized cross-over trial. Two trials compared potassium chloride (KCl) to potassium citrate (K-cit), one trial without a placebo group. Potassium citrate is the form most similar to that provided naturally in food. Six studies were judged as positive quality and four were neutral in quality.

Enrollment criteria differed among the studies. Three systematic review/meta-analyses included trials that studied hypertensive or normotensive populations, or both. One systematic review/meta-analysis and one RCT studied just hypertensive subjects. Three RCTs and one non-randomized trial enrolled non-hypertensive individuals. The trials were conducted in China, New Zealand, Okinawa and the United Kingdom. Two systematic review/meta-analysis included studies from Australia, Germany, Italy, Japan, Kenya, Netherlands, New Zealand, the United Kingdom and the US; two did not identify the study locations. The five systematic reviews/meta-analyses had some overlap between included trials; this ranged from five out of five (Dickinson et al, 2006) to 25 out of 55 (Burgess et al, 1999).

Each study reported the effects of potassium intake, either from supplements or diet, on blood pressure (BP) in adults. Considering all studies, there was a significant reduction in either systolic blood pressure (SBP) or diastolic blood pressure (DBP) in all but one study and significant reductions in both SBP and DBP in four studies. Among the four RCTs studies judged to be of positive quality, there was a significant reduction in SBP or DBP in each study.

Three meta-analyses of these trials document that, on average, increased potassium intake lowers BP (Cappuccio and MacGregor, 1991; Geleijnse, 2003; Whelton, 1997). In the meta-analysis by Whelton et al, (1997), average net SBP/DBP reductions from a net increase in urinary potassium excretion of 2g per day (50mmol per day) were 4.4/2.5mmHg among hypertensive individuals and 1.8/1.0mmHg among non-hypertensive individuals. A meta-analysis (Dickinson et al, 2006) did not detect a significant effect of potassium on BP, but this meta-analysis applied especially restrictive exclusion criteria (just hypertensive individuals, with at least eight weeks of treatment) and included only five trials. The review by Burgess (1999) was not a formal meta-analysis. These BP reductions tended to be greatest in hypertensive individuals and Blacks.

Mostly on the basis of literature reviewed for the Dietary Guidelines 2005 (DG2005), we conclude that increased potassium intake lowers BP (Evidence Level: Strong).

Evidence summary paragraphs

Systematic Reviews/Meta-analyses

Burgess E et al, 1999 (neutral quality) a systematic review of 55 studies was conducted to update evidence-based Canadian recommendations for dietary or supplemental cation intake, including potassium, for the prevention and treatment of HTN in otherwise healthy adults (except pregnant women). MEDLINE was searched for reviews, meta-analyses, observational studies and RCTs published in English or French from 1966 to 1996. Included health outcomes were changes in BP, morbidity and mortality. Articles were reviewed, classified according to study design and graded using Canadian Hypertension Society principles. Higher value was placed on the prevention of cardiovascular morbidity and premature death from untreated HTN. The panel concluded that the evidence did not support potassium supplementation to prevent BP increases in normotensives or to reduce BP in hypertensives. The panel did find that potassium supplementation may be effective in reducing BP in hypokalemic patients during diuretic therapy. The panel recommended that, for prevention of HTN and reduced risk of stroke-related mortality, daily potassium intake should be 60mmol or more through dietary intake, not supplementation.

Cappuccio F et al, 1990 (positive quality) a meta-analysis conducted in England, analyzed 19 clinical trials that studied the effect of potassium supplementation on BP. The studies included a total of 586 participants; 412 with essential hypertension. Pooled analysis of the effect of potassium supplementation estimated a 5.9mmHg reduction in SBP (95% CI: -6.6 to -5.2mmHg) and 3.4mmHg reduction in DBP (95% CI: -4.0 to 2.8mmHg). Analysis of only hypertensive subjects found that the magnitude of the blood pressure lowering effect was greater. Systolic blood pressure decreased 8.2mmHg (95%CI: -9.1 to -7.3mmHg) and DBP decreased 4.5mmHg (95%CI: -5.2 to -3.8mmHg). Weighted regression analysis showed a significant relationship between decrease in BP and the duration of supplementation ($P<0.05$ and $P<0.01$ for SBP and DBP, respectively). The authors concluded that non-pharmacological approaches for BP control in subjects with uncomplicated essential HTN should include a recommendation for increased potassium intake.

Dickinson et al, 2006 (positive quality), a Cochrane systematic review and meta-analysis of trials conducted in the US, Australia, Kenya, Germany and Italy, evaluated the effects of potassium supplementation on health outcomes and BP in adults with HTN. One of the exclusion criteria was treatment duration of at least eight weeks. After exclusion criteria were applied, five RCTs of parallel or crossover design that compared oral potassium supplements with placebo, usual care, or no treatment were identified that included a pooled total of 425 participants whose SBP was >140 mmHg and DBP was >85 mmHg without a known primary cause. Compared to control subjects, potassium supplementation resulted in a statistically non-significant reduction in SBP (mean difference: -11.2, 95% CI: -25.2 to 2.7) and DBP (mean difference: -5.0 (95% CI: -12.5 to 2.4). Excluding one trial in an African population with very high baseline BP resulted in small overall reductions in BP (SBP mean difference= -3.9, 95% CI: -19.9, 5.7; DBP mean difference= -5.5, 95% CI: -14.5 to 3.5). Two trials administering lower (fewer doses of potassium showed greater reductions in BP than three trials administering >100 mmol per day, which was significant for DBP (mean differences in the two trials= -17.00 (95% CI: -19.25, -14.75) and -10.50 (95%CI: -16.32, -4.68)mmHg). While the authors concluded the effect of potassium supplementation on BP is uncertain, this meta-analysis applied especially restrictive exclusion criteria.

Geleijnse et al, 2003 (neutral quality) a meta-analysis, conducted in the Netherlands, examined RCTs to assess BP response to sodium and potassium intake in adults. MEDLINE (January 1995-March 2001) and reference lists of systematic reviews were searched and 27 trials (N=30 strata) met the criteria for review, which included a minimum duration of two weeks. Data on changes in electrolyte intake and BP during intervention were collected, along with data on mean age, gender, body weight, initial electrolyte intake and initial BP of the study populations. Weighted meta-regression was used to analyze the effect of potassium intake on BP. Analyses were repeated with adjustment for potential confounders. Increased potassium intake (median: 44 mmol per 24 hours) resulted in a 2.42mmHg decrease in SBP (95% CI:-3.75, -1.08mmHg) and a 1.57mmHg decrease in DBP (95%CI:-2.65, -0.50). Blood pressure response was larger in hypertensives than normotensives, (SBP: -3.51 vs. -0.97mmHg, P=0.089; DBP: -2.51 vs. -0.34mmHg, P=0.074). The authors concluded that increasing potassium consumption could help reduce BP, especially in populations with HTN.

Whelton P et al, 1997 (positive quality) a meta-analysis, conducted in the US, examined 33 RCTs to assess the effect of potassium supplementation on BP in adults (pooled N=2,609 participants). Included studies were published before July 1995 in the English language. A standardized protocol was used to extract information on sample size, duration, study design, potassium dose, participant characteristics and treatment results. Each trial's results were weighted by the inverse of its variance, then a random-effects model was used to pool findings. One trial was excluded because an extreme BP lowering effect was noted. After exclusion, pooled analysis found that potassium supplementation was associated with a 3.11 mmHg reduction in mean SBP (95% CI:-1.91 to -4.31mmHg) and 1.97mmHg reduction in mean DBP (95% CI: -0.52 to -3.42mmHg), both P<0.001. It was also reported that the effects of treatment appeared to be enhanced in studies in which participants were concurrently exposed to a high intake of sodium. The authors concluded that low potassium intakes may contribute to the onset of high BP and that increased potassium intake should be considered as a recommendation for prevention and treatment of HTN, especially for individuals who are unable to reduce their sodium intake.

Randomized Controlled Trials

Braschi et al, 2008 (positive quality) a randomized, double-blind placebo-controlled trial conducted in the United Kingdom, compared the effect of supplementation with KCl or K-cit on BP in predominately healthy, normotensive volunteers (N=90, age range 33.8 to 36.9 years, body mass index (BMI) across groups: 22.55 to 25.2kg/m². After a two week run-in, subjects were randomly assigned to receive KCl (30mmol K), K-cit (30mmol K) or placebo for six weeks. Urinary electrolyte excretion, plasma electrolytes, BP, BMI and heart rate were measured at baseline and end of intervention. At baseline, mean K excretion was over 70mmol per day in each group. Compared with placebo, KCL significantly reduced SBP, DBP and mean arterial pressure (MAP) by 5.2, 4.3 and 4.7mmHg respectively. Corresponding reductions for K-citrate were 6.7, 4.3 and 5.2mmHg (all statistically significant). The BP changes induced by K-cit and KCl were NS different from each other, and were not related to baseline urinary electrolytes. There was a greater treatment-related effect observed in those with higher baseline SBP (P=0.007). This study documented that a modest dose of potassium, only 30mmol per day, reduces blood pressure. Even though there was no significant difference between the KCL and K-Cit groups, the study was likely underpowered to detect a difference in BP.

He et al, 2005 (positive quality), a randomized cross-over trial conducted in the United Kingdom, compared the effects of supplementation with KCl (96mmol K) and K-cit (96mmol K) on BP in 14

hypertensive Caucasian adults (11 males, three females; mean age: 51+9 years; baseline BP=151+16/93+7mmHg; 24-hour urinary K=164+36mmol). Subjects were randomly assigned to first receive KCl or K-cit for one week, followed by the other intervention, with a one-week washout period in between. Blood pressure, body weight and plasma and urinary electrolytes were measured at baseline and at the end of each treatment period. After KCl, BP was 140+12/88+7mmHg (SBP: P<0.001, DBP: P<0.01 compared to baseline) and urinary K was 164+36mmol. After K-cit, BP was 138+12/88+66mmHg (SBP: P<0.01, DBP: P<0.05 compared to baseline) and urinary K was 160+33mmol. There were NS differences in BP between treatments. Plasma K values were significantly higher after both interventions (KCl: 4.6+0.3mmol per L; K-cit: 4.6+0.3mmol per L) than at baseline (4.2+0.3mmol per L) (P<0.001). Study limitations include the small number of subjects and the lack of a placebo group. Overall, the authors concluded that KCl and K-cit have a similar effect on BP. Even though there was NS difference between KCL and K-Cit, the study was likely underpowered to detect a difference in BP.

He J et al, 2009 (positive quality). This non-randomized, controlled three-week feeding trial, conducted in rural China, examined gender differences in BP response to dietary sodium and potassium intake. The interventions included seven days on a low-sodium diet (51.3mmol per day), seven days on a high-sodium diet (307.8mmol per day) followed by seven days on a high-sodium (307.8mmol per day) plus potassium supplementation (60mmol per day), with no washout period between interventions. Subjects were 1,906 adults (1,010 men and 896 women), SBP 130-160 and DBP 85-100mmHg; including eligible siblings and offspring, aged 18-60 years, who participated in the Genetic Epidemiology Network of Salt Sensitivity (GenSalt) study. During the interventions, meals were prepared without salt. Staff added prepackaged salt to individual meals prior to serving and observed subjects' consumption. Food records were kept for each meal. At baseline and in each phase of the intervention, three timed urine specimens were collected (one 24-hour and two overnight) to assess dietary compliance. Nine BP measurements were obtained during the three-day baseline observation and the last three days of each intervention using a random-zero sphygmomanometer. For an assessment of the effects of potassium on BP, only the last two periods are relevant (high salt/low potassium, compared to high salt/high potassium). In men, potassium lowered SBP by 4.4 and DBP by 1.5mmHg (each statistically significant). In women, potassium also lowered BP (SBP reduction of -4.5 and DBP of -2.1mmHg); the DBP reduction in women was slightly, but significantly higher than that observed in the mean (P=0.007). Study strengths include excellent compliance, inclusion of an arm with increased potassium, rigorous methods and conduct of a trial in an understudied, non-overweight population. Limitations include the short duration of each intervention phase (seven days), lack of a washout period, non-randomized order of diets, pre-post evaluation and single ethnic group (rural Chinese). Overall, results from this trial suggest that an increased intake of potassium lowers BP in a pre-hypertensive and hypertensive generally lean, Asian population.






Hilary Green et al, 2000 (neutral quality), a randomized crossover double-blind controlled trial conducted in New Zealand, evaluated the effect of high-calcium skim milk or potassium-enriched high-calcium skim milk on BP compared with non-enriched skim milk. Healthy subjects (N=19 males, mean age=55+11 years, BMI=27.0+2.3kg/m²; 19 females, mean age=50+10 years, BMI=25.7+4.6kg/m²) who were not receiving pharmacologic treatment for HTN, replaced their usual liquid milk with two servings (480ml) per day of reconstituted skim milk powder (SMP) (720mg Ca, 885mg K, 64mg Mg, 197mg Na), high-calcium SMP (1,075mg Ca, 855mg K, 75mg Mg, 208mg Na) or potassium-enriched high-calcium SMP (1,040 Ca, 1,585mg K, 71mg Mg, 195mg Na) for four weeks each, in randomized order, with a four-week washout periods between each milk intervention. Sitting and standing BP were recorded at baseline and at weeks two and four of each intervention. Ambulatory BP (eight hours) was also recorded at the start and end of each





intervention. Changes in body weight, physical activity and food intake were monitored; there were NS changes during the study. The authors reported only pre-post changes and do not perform between-group statistical tests. The potassium-enriched high-calcium milk intervention decreased sitting SBP from 125+18 to 117+16mmHg ($P<0.001$) and ambulatory SBP (138+13 to 135+11mmHg, $P<0.05$) and DBP (80+8 to 78+9mmHg, $P<0.05$). Standing SBP decreased on each of the milk interventions: SMP: 127+16 to 124+16mmHg; high-calcium SMP: 130+18 to 126+17mmHg ($P<0.05$), potassium-enriched high-calcium SMP: 130+16 to 122+15mmHg ($P<0.05$). The authors conclude that increased calcium and potassium may help to prevent the development of HTN. However, the lack of between-group statistical testing is a major limitation which makes it impossible to reach this conclusion.

Tuekpe et al, 2006 (neutral quality), an RCT conducted in Okinawa, examined whether increasing the consumption of the yellow-green Okinawan vegetables used in Okinawan dishes increases potassium intake. Subjects ($N=39$ normotensive, normal weight females; 20-30 years old) were randomly assigned to receive an average weight of 371.4g per day of five typical yellow green Okinawan vegetables (2.6kg per week, delivered twice weekly) for 14 days. Urinary potassium was collected via 24-hour urine sample pre- and post-intervention. The intervention group consumed an average of 144.9g of Okinawan vegetables per day. Urinary potassium excretion increased significantly (363.5mg per day, $P=0.047$) from pre- to post-intervention in the intervention group with NS changes in the control group. Post-intervention urinary potassium excretion correlated positively with vegetable consumption in both the intervention ($r=0.79$, $P<0.0001$) and control ($r=0.58$, $P=0.008$) groups, as well as with the Okinawan vegetable intake in the intervention group ($r=0.73$, $P=0.0004$). Changes in BP were NS; all subjects were normotensive and of normal weight. The authors found that increasing the consumption of yellow-green Okinawan vegetables typically significantly increased urinary potassium, a reflection of increased potassium intake. The trial was likely underpowered to detect a difference in BP.

 [View table in new window](#)

Author, Year, Study Design, Class, Rating	Population	Significant Systolic Blood Pressure Reduction	Significant Diastolic Blood Pressure Reduction	Caveats
Braschi A and Naismith DJ 2008 Study Design: Randomized, doubleblind placebo-controlled trial with parallel arm design. Class: A	Normotensive.	+	+	None.

Rating: 				
<p>Burgess E, Lewanczuk R et al, 1999</p> <p>Study Design: Review (panel)</p> <p>Class: R</p> <p>Rating: </p>	<p>Hypertensive and Normotensive (not pooled).</p> <p>Systematic Review 55 trials (18 epi; 37 RCT).</p>	∅	∅	None.
<p>Cappuccio F and MacGregor G, 1991</p> <p>Study Design: Meta-analysis</p> <p>Class: M</p> <p>Rating: </p>	<p>Hypertensive and Normotensive; N=586.</p> <p>Systematic Review/Meta-analysis 19 RCTs (13 HTN, Six NTN).</p>	+	+	<p>SBP: P<0.05.</p> <p>DBP: P<0.01.</p>
<p>Dickinson HO et al 2006</p> <p>Study Design: Systematic Review/Meta-analysis</p> <p>Class: M</p> <p>Rating: </p>	<p>Hypertensive.</p> <p>N=425.</p> <p>Systematic Review/Meta-analysis five RCT.</p>	∅	+ (two trials).	None.
<p>Geleijnse JM, Kok FJ et al, 2003</p> <p>Study Design: Meta-analysis</p> <p>Class: M</p> <p>Rating: </p>	<p>N=30 strata.</p> <p>Meta-analysis (27 RCT for K).</p>	+	+	<p>SBP: P=0.089.</p> <p>DBP: P=0.074.</p> <p>BP response greater in HTN subjects.</p>

<p>He FJ et al 2005</p> <p>Study Design: Randomized cross-over trial</p> <p>Class: A</p> <p>Rating: </p>	Hypertensive.	+	+	Small sample size, N=14.
<p>He J, Gu D et al, 2009</p> <p>Study Design: Non-randomized trial</p> <p>Class: M</p> <p>Rating: </p>	Normotensive.	+ (Age and Baseline BP). Ø (Gender).	+ (Gender and Baseline BP). Ø (Age).	None.
<p>Hilary Green J, Richards JK, and Bunning RL 2000</p> <p>Study Design: Randomized double blind controlled trial</p> <p>Class: A</p> <p>Rating: </p>	Normotensive.	+	+	Δs in office DBP were NS. Ambulatory BP was significant.
<p>Tuekpe MK et al 2006</p> <p>Study Design: Randomized Controlled Trial</p> <p>Class: A</p> <p>Rating: </p>	Normotensive.	Ø	Ø	Study designed to examine urinary K excretion, not BP.
<p>Whelton, Appel et al, 1998</p> <p>Study Design: Randomized controlled study</p>	<p>N=2,609; 44 evaluated two times in separate protocols.</p> <p>Meta-analysis (33 RCTs).</p>	+	+	SBP and DBP: P<0.001.


Class: A				
Rating: 				





Research Design and Implementation Rating Summary

For a summary of the Research Design and Implementation Rating results, [click here](#).


Worksheets


 [Braschi A, Naismith DJ. The effect of a dietary supplement of potassium chloride or potassium citrate on blood pressure in predominantly normotensive volunteers.. Br J Nutr. 2008 ;99\(6\):1284-92.](#)


 [Burgess E, Lewanczuk R, Bolli P, Chockalingam A, Cutler H, Taylor G, Hamet P. Lifestyle modifications to prevent and control hypertension. 6. Recommendations on potassium, magnesium and calcium. Canadian Hypertension Society, Canadian Coalition for High Blood Pressure Prevention and Control, Laboratory Centre for Disease Control at Health Canada, Heart and Stroke Foundation of Canada. CMAJ. 1999 May 4; 160 \(9 Suppl\): S35-S45.](#)

 [Cappuccio FP, MacGregor GA. Does potassium supplementation lower blood pressure? A meta-analysis of published trials. J Hypertens. 1991 May; 9 \(5\): 465-473.](#)


 [Dickinson HO, Nicolson DJ, Campbell F, Beyer FR, Mason J. Potassium supplementation for the management of primary hypertension in adults. Cochrane Database Syst Rev. 2006 Jul 19;3.](#)

 [Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: A metaregression analysis of randomised trials. J Hum Hypertens. 2003 Jul; 17 \(7\): 471-480.](#)

 [He FJ, Markandu ND, Coltart R, Barron J, MacGregor GA. Effect of short-term supplementation of potassium chloride and potassium citrate on blood pressure in hypertensives.. Hypertension. 2005 Apr;45\(4\):571-4.](#)

 [He J, Gu D, Chen J, Jaquish CE, Rao DC, Hixson JE, Chen JC, Duan X, Huang JF, Chen CS, Kelly TN, Bazzano LA, Whelton PK; GenSalt Collaborative Research Group. Gender difference in blood pressure responses to dietary sodium intervention in the GenSalt study. J Hypertens. 2009 Jan; 27\(1\): 48-54.](#)

 [Hilary Green J, Richards JK, Bunning RL. Blood pressure responses to high-calcium skim milk and potassium-enriched high-calcium skim milk. J Hypertens. 2000 Sep;18\(9\):1331-9.](#)

 [Tuekpe MK, Todoriki H, Sasaki S, Zheng KC, Ariizumi M. Potassium excretion in healthy Japanese women was increased by a dietary intervention utilizing home-parcel delivery of Okinawan vegetables. Hypertens Res. 2006;6:389-396.](#)

 [Whelton PK, Appel LJ, Espeland MA, Applegate WB, Ettinger WH, Kostis JB, Kumanyika S, Lacy CR, Johnson KC, Folmar S, Culter JA. Sodium Reduction and Weight Loss in the](#)

Treatment of Hypertension in Older Persons; A Randomized Controlled Trial of
Nonpharmacologic Interventions in the Elderly (TONE). TONE Collaborative Research Group.
JAMA 1998 Mar 18; 279 (11): 839-846. Erratum in: *JAMA* 1998 Jun, 24; 279 (24): 1954